

# Chlamydia trachomatis as a possible cause of peritonitis and perihepatitis in a young woman

J PAAVONEN\* AND V V VALTONEN†

From the \*I-II Departments of Obstetrics and Gynaecology, University Central Hospital, Helsinki, and the †Department of Serology and Bacteriology, University of Helsinki, Finland

**SUMMARY** In a 20-year-old woman who developed pelvic peritonitis and perihepatitis, *Chlamydia trachomatis* was isolated from the lower genital tract. There was a significant rise in the antibody titre against *C trachomatis*, suggesting that an ascending infection by this organism may have been responsible for the condition.

## Introduction

The syndrome of perihepatitis is not a rare complication of gonococcal pelvic inflammatory disease (PID).<sup>1,2</sup> Perihepatitis associated with PID is also known as the Fitz-Hugh-Curtis syndrome.<sup>3,4</sup> Recently, Müller-Schoop *et al*<sup>5</sup> found serological evidence that *Chlamydia trachomatis* in the absence of gonococcal infection may play an important role in this peritoneal inflammation. A case of PID due to *C trachomatis* with exudative ascites and pain and tenderness in the right upper quadrant suggestive of perihepatitis in a young woman is presented.

## Case report

A 20-year-old woman was admitted to hospital because of pelvic pain. The patient was para 1 with no miscarriages. She had previously been well with a regular menstrual cycle.

## PAST HISTORY

She had had a normal delivery eight months previously; during pregnancy she had been examined at the outpatient clinic several times because of lower abdominal pain. Laboratory examination had shown pyuria without significant bacteriuria on three separate occasions. At 21 weeks of pregnancy she had been treated with nitrofurantoin 50 mg three times daily for three weeks. Cultures for *C trachomatis* were not performed at that time. An intrauterine device (IUD) was inserted five months after delivery.

Address for reprints: Dr J Paavonen, I-II Departments of Obstetrics and Gynaecology, University Central Hospital, Haartmaninkatu 2, SF-00290 Helsinki 29, Finland

Received for publication 15 January 1980

## PRESENT HISTORY

The patient's last menstrual period had occurred two weeks before admission. One week later she had had lower abdominal cramp and increased vaginal discharge. She was then admitted to hospital by her family doctor because of suspected upper genital tract infection.

On admission a gynaecologist found purulent cervicitis and adnexal tenderness on both sides. Her temperature was 36.8°C. White blood cell (WBC) count was  $8.0 \times 10^9/l$ . A Papanicolaou cervicovaginal smear showed severe inflammatory changes (class II) and a large number of leucocytes. Cervical cultures for *Neisseria gonorrhoeae*, *Trichomonas vaginalis*, and yeasts all gave negative results. Cultures for *C trachomatis* were performed simultaneously using an isolation technique described elsewhere.<sup>6</sup> The patient was discharged and treated for suspected PID with penicillin V 2 megaunits orally four times daily and metronidazole 300 mg orally three times daily.

Four days later however the patient was readmitted because of vomiting and pelvic and upper abdominal pain. Her temperature was 37.8°C. Examination showed pelvic peritonitis and severe tenderness at the right costal margin (Murphy's sign). The WBC count was  $10.5 \times 10^9/l$ , and the erythrocyte sedimentation rate (ESR) was 20 mm in the first hour. Culdocentesis produced 400 ml of yellowish serous inoffensive fluid. Culture results for *C trachomatis* from both cervical and urethral specimens were positive. Parenteral treatment with doxycycline 100 mg 12 hourly for four days was started (and continued orally for 10 days). Acute-phase titre in the single-antigen immunofluorescence test (IFAT) for IgG antichlamydial antibodies<sup>7</sup> was 128. Radiographs of the chest and abdomen were normal.

On examination the following day the patient appeared well. Her temperature was normal and no pelvic mass could be found. However, Murphy's sign was still strongly positive. Her IUD was then removed; there was no growth of aerobic or anaerobic bacteria on culture. Urine culture for bacteria gave a negative result. Serum urea, serum and urine amylases, serum electrolytes, and serum transaminases (on four consecutive days) were all within normal limits. Cultures of stool specimens on three consecutive days gave negative results for yersinia, salmonellae, and shigellae. Blood culture for bacteria gave repeatedly negative results. Culture of the culdocentesis aspirate yielded a *Peptostreptococcus*, sensitive in vitro to penicillin and metronidazole but resistant to tetracycline; the fluid was not cultured for chlamydia. Gynaecological examination five days later was normal. The ESR was 25 mm in the first hour, WBC count,  $5.9 \times 10^9/l$ , and the IFAT titre for antichlamydial antibodies on serum obtained nine days after the first specimen had risen to 1024 (an eightfold increase). The patient was discharged on the tenth day after admission. Follow-up examination showed no abnormalities and the patient was asymptomatic.

## Discussion

Except by laparoscopy, there is no specific test for diagnosing perihepatitis. Severe right upper abdominal pain and tenderness may be indistinguishable from acute cholecystitis.<sup>8</sup> It has been previously estimated that about 3% of patients with signs and symptoms of acute cholecystitis actually have gonococcal perihepatitis.<sup>8</sup> On the other hand, the incidence of gonococcal perihepatitis has been estimated to be less than 10% in women with gonococcal infection.<sup>9</sup> *C. trachomatis* has been implicated as an important cause of non-specific genital infections, which are at present a major health problem.<sup>10, 11</sup>

Recent results have provided strong support for the view that acute salpingitis may, in many cases, be caused by *C. trachomatis*.<sup>12-14</sup> In this case, chlamydial infection was confirmed by positive culture results from cervical and urethral specimens. Furthermore, high antichlamydial antibody titres with a significant rise between the acute and convalescent phases suggested that *C. trachomatis* actively participated in this systemic disease. Concomitant infection with *N. gonorrhoeae* was definitely excluded by negative culture results. Further support for the aetiological role of *C. trachomatis* was obtained from the poor response to the initial treatment with penicillin and metronidazole, both of which are known to be ineffective against chlamydia. However, when

doxycycline treatment was started the patient rapidly responded clinically. Acute cholecystitis was an unlikely cause of the patient's symptoms as consecutive liver function tests showed normal results.

The fact that culture of the culdocentesis aspirate yielded *Peptostreptococcus* is interesting and raises the question of its possible aetiological role in the disease. Together with the modern techniques of anaerobic bacteriology, studies using culdocentesis have suggested that obligate anaerobes often play a part in the aetiology of acute PID.<sup>15</sup> However, caution should be used in interpreting the bacteriological results from specimens obtained by culdocentesis because of possible contamination of the specimens by micro-organisms from the vaginal flora.<sup>16</sup> In this case the *Peptostreptococcus* isolated was sensitive to penicillin and metronidazole in vitro, but this combined treatment was ineffective clinically, thus not supporting its aetiological role.

It is still uncertain how the infection reaches the peritoneum and the subphrenic space. A small amount of exudative fluid is not uncommon with PID and is thought to be produced by the inflamed tubal and ovarian tissue.<sup>17</sup> The precise aetiology of ascites, or overproduction of fluid within the peritoneal cavity, is difficult to establish. However, it may result from the rapid spread of a genital tract infection to the peritoneum, which—secondary to inflammation and adhesion formation—blocks the normal lymphatic pathways for excretion of fluid from the cavity.<sup>18</sup> Thus perihepatic inflammation is most likely caused by infection which has ascended from the pelvis.

In conclusion, *C. trachomatis*, a prevalent sexually transmitted organism, may be a probable cause of exudative peritonitis and perihepatitis as a complication of acute PID. This syndrome has earlier been related only to gonococcal PID. This report may encourage clinicians to look for evidence of chlamydial infection in similar cases.

## References

1. Litt IF, Cohen MI. Perihepatitis associated with salpingitis in adolescents. *JAMA* 1978; **240**: 1253-4.
2. Semchyschyn S. Fitz-Hugh-Curtis syndrome. *J Reprod Med* 1979; **22**: 45-8.
3. Curtis AH. A cause of adhesions in the right upper quadrant. *JAMA* 1930; **94**: 1221-2.
4. Fitz-Hugh T Jr. Acute gonococcal peritonitis of the right upper quadrant in women. *JAMA* 1934; **102**: 2094-6.
5. Müller-Schoop JW, Wang SP, Munzinger J, Schlöpfer HU, Knoblauch M, Amman RW. *Chlamydia trachomatis* as a possible cause of peritonitis and perihepatitis in young women. *Br Med J* 1978; **i**: 1022-4.
6. Paavonen J, Saikku P, Vesterinen E, Lehtovirta P. Infertility and cervical *Chlamydia trachomatis* infections. *Acta Obstet Gynecol Scand* 1979; **58**: 301-3.

7. Saikku P, Paavonen J. Single-antigen immunofluorescence test for chlamydial antibodies. *J Clin Microbiol* 1978; **8**: 119-21.
8. Knorrning J, Nieminen J. Gonococcal perihepatitis in a surgical ward. *Ann Clin Res* 1979; **11**: 66-70.
9. Stanley MM. Gonococcal peritonitis of the upper part of the abdomen in young women. *Arch Intern Med* 1946; **78**: 1-13.
10. Anonymous. Non-specific genital infection. *Br Med J* 1979; **ii**: 161-2.
11. Paavonen J. Chlamydial infections. Microbiological, clinical and diagnostic aspects. *Med Biol* 1979; **57**: 135-51.
12. Mårdh P-A, Ripa T, Svensson L, Weström L. *Chlamydia trachomatis* infection in patients with acute salpingitis. *N Engl J Med* 1977; **296**: 1377-9.
13. Trehan JD, Ripa KT, Mårdh P-A, Svensson L, Weström L, Darougar S. Antibodies to *Chlamydia trachomatis* in acute salpingitis. *Br J Vener Dis* 1979; **55**: 26-9.
14. Paavonen J, Saikku P, Vesterinen E, Aho K. *Chlamydia trachomatis* in acute salpingitis. *Br J Vener Dis* 1979; **55**: 203-6.
15. Eschenbach DA, Buchanan TM, Pollock HM, et al. Polymicrobial etiology of acute pelvic inflammatory disease. *N Engl J Med* 1975; **293**: 166-71.
16. Cunningham FG, Hauth JC, Gilstrap LC, Herbert WNP, Kappus SS. The bacterial pathogenesis of acute pelvic inflammatory disease. *Obstet Gynecol* 1978; **52**: 161-4.
17. Plentl AA, Friedman EA. *Lymphatic System of the Female Genitalia*. Philadelphia: WB Saunders Co, 1971.
18. Berek JS, Darney PD. Massive exudative ascites produced from a tubal pseudocyst in chronic pelvic inflammatory disease. *Obstet Gynecol* 1979; **54**: 490-2.